MEDICAL EMERGENCIES

I.P. Fairbairn, Clinical Lecturer and Honorary Specialist Registrar in Respiratory Medicine, Royal Infirmary of Edinburgh

SESSION 1

MEDICAL EMERGENCIES: IMPROVING THE PROCESS OF CARE

Chaired by Dr Derek Bell, Patient Services Director, Royal Infirmary of Edinburgh

Who should be seen and when? Current proposals by the College

Dr Mary Armitage, Consultant Endocrinologist, Royal Bournemouth Hospital

Dr Armitage referred to three working party reports: *Acute Medical Admissions and the Future of General Medicine*,[1] *Acute Medicine: the Physician's Role*[2] and *Governance in Acute Medicine*.[3]

The main problems facing acute medicine are the rise in emergency admissions and the lack of an adequate infrastructure – with too few beds and too few trained staff. These problems are all the more acute in a service that needs to operate around the clock. One area where improvements could be made is the interface between the Accident and Emergency Department (A&E) and the acute medical unit.

The number of hospital admissions have risen year-on-year every year since the birth of the NHS, while the total number of NHS beds has been reduced. To partially compensate for this year-on-year increase, there has been an increase in nursing home beds and thus the total bed stock has remained reasonably static. In addition, the length of in-patient stay has been dropping. In 1949, a patient with a myocardial infarction (MI) had an average hospital stay of 49 days, compared with five days today! This means that throughput has increased, with increased pressures being brought to bear on medical and nursing staff.

More recent statistics are a cause of some concern. A joint working party from the Department of Health and the Royal College of Physicians (RCP) (London) reporting on 12 December 2001 noted that the average length of hospital stay plateaued in 1999 but rose in 2001, largely because of an increased duration of stay in the elderly. A further major contributing factor is likely to be that the number of nursing home beds has now started to fall.

With the aim of improving efficiency, moves to increase workforce flexibility have resulted in employees working across professional boundaries. This has led to the questions of who should be seen, when, where, and by whom.

The natural expectation of patients is to see a senior doctor when they are at their sickest. In addition, evidence has accumulated that specialist care for a number of illnesses (e.g. asthma, gastrointestinal (GI) bleeding) results in improved outcome. This has led to a conflict between generalists and specialists. The challenge, therefore, is to deliver the benefit of specialist care to patients whilst maintaining high-quality acute medicine.

The RCP London recommends a broad base of physicians (trained in acute medicine as well as a specialty), who are dually accredited and practising both sub-specialties. In addition, specialist triage should be provided, through which patients directly benefit from immediate specialist care.

Each year, 13 million people attend A&E in England and Wales, but only four million are admitted. Three million patients dial 999, one million are referred by their GP and nine million self-refer. The route of admission may influence the quality of care: in order to address this the concept of an 'integrated assessment of emergency admissions' has been mooted. This aims for care to be needs-related, irrespective of route of referral. To achieve this, A&E and acute medicine need to function as a continuum, with a seamless journey for the patient between the two.

In more practical terms, medical emergencies need access to an initial assessment, resuscitation, acute medical care, intensive care, high-dependency care, coronary care and acute trauma/surgical care. Some patient groups need fast-tracked care, e.g. MIs requiring thrombolysis. However, other patients could avoid attending A&E altogether if it were possible to improve: primary care access to investigations; direct access to social services and community facilities; emergency out-patient clinics; rapid access chest pain service; and rapid multidisciplinary assessment for patients with complex needs.

Problems facing a patient who requires an acute general medical assessment include delays, indiscriminate re-clerking and poor clinical record keeping. To improve this, the medical Royal Colleges recommended a single, structured, generic clerking record for both A&E and acute medicine.

Different hospitals assess their acute medical admissions in different surroundings: a medical assessment unit; a medical admissions unit; an observation area; an 'alpha' ward; or general medical/surgical wards. Whatever model is used, the Colleges recommend that there needs to be a single area for the initial assessment of acutely ill patients. This enables regular observations and review and follow-up through regular post-take ward rounds. A sufficient number of staff with the correct skill base and operating extended roles can be concentrated in this area. The appropriate access to investigations and discharge planning must also be in place.

The RCP (London) recommends that, 'at all times there is a consultant physician available to give clinical leadership for acute medical care'.[3] Ultimately, it is hoped that 'available' means a round-the-clock presence. Obviously, this responsibility could not rest on one individual and the consultant could be the 'on-take' physician or an acute care physician. The RCP recommends that, 'acute trusts consider how this leadership should be provided – considering local circumstances and arrangements at the A&E/acute medicine interface' and that, 'trusts consider [the] appointment of a physician as clinical director of acute medical services, to manage and organise systems and working practices at the A&E/acute medicine interface'.[3]

Other recommendations concern training in acute medicine. Ideally, general professional training should include attachments in A&E and intensive care medicine. Higher medical training should reflect clinical practice and include time for acute general medicine. Other practical recommendations are permanent access to old notes and to laboratory investigations and support services. Audit of acute medicine services and clinical protocols for acute illnesses are also encouraged.

Waits and delays: a thing of the past?

Ms Karen Castille, Director, Emergency Services Programmes, NHS Modernisation Agency, London

Ms Castille described the IDEA programme, which aims to apply modern operations management techniques to reduce waits and delays in emergency care. Problems with the current system include: access confusion; repetition; 'hand-offs' (with patients being passed from one specialty to another); pushing from one queue to another; fast-tracking to the detriment of others; delayed diagnostics; batching of tests; and waits and delays across the whole system. IDEA entails improving flow and throughput. To achieve this, it utilises a number of manufacturing principles. This has led to the idea of redefining patient flows.

Currently, acute patients who present to hospital are triaged, have a fuller history taken, have laboratory or radiological tests, are reviewed, have further tests, have treatment, get better before finally being discharged. However, the route and speed at which different patient groups travel through this system varies greatly. By trying to force all patients through the same pathway, a flow mayhem is created and flow rates are compromised. This results in a backlog of patients and frequently obscures the actual source of the bottleneck.

Streaming, separating service, flow management and segmentation are all 'management speak' for the same principle. It considers the whole patient journey and groups similar process requirements and flow rates. It aims to match capacity to demand, improving flow and keeping it constant, thus resulting in reductions in delays and waiting times for all patients.

Flows can be grouped together in various ways: volume (runners), time (minors vs majors), clinical (abdominal pain, chest pain, elderly falls etc.) and chronological flow processes. Potential emergency flows can range from assessment–advice–discharge to assessment–resuscitation–institution and investigations–treatment–admit–discharge. By identifying the various groups of patients and their different potential flows, the patient's visit can be streamlined, resulting in easier processing and improved teamwork.

The introduction of segmentation in Kettering increased the number of patients treated within one hour from 52% to 75%.

Who should see the patient first?

Dr Elizabeth Myers, Nurse Consultant for Acute Receiving, Tayside University Hospitals, Dundee

Dr Myers discussed the role of non-medical staff, and in particular nurse consultants, in the assessment and treatment of emergency patients. In explaining what they did, she referred to the core elements of their role: expert practice; professional leadership; education, training and development; and practice and service development, research and evaluation. At Dundee, she had changed the service in three areas: GP–nurse referral; triage nursing; and nurse assessment.

Prior to these changes, medical registrars were unable to assess patients efficiently because of continuous interruptions; their performances differed depending on their experience and knowledge of local systems. General practitioners preferred referring to nurses because of shorter response times and because the nurse was more focused on thinking ahead to the discharge.

Nurse assessment at Dundee is going to be targeted at patients with diagnostic uncertainty. Nurses will take a history, examine and order investigations according to a protocol. Four potential areas are DVT, pleuritic chest pain, exacerbations of COPD and poor mobility in the elderly.

SESSION 2

ACUTE CORONARY SYNDROMES: TREATMENT STARTS AND FINISHES AT HOME

Chaired by Professor Keith A.A. Fox, University of Edinburgh

Thrombolysis should begin at home

Mr James Ferguson, Consultant in A&E Medicine, Aberdeen Royal Infirmary

The benefit of early thrombolysis for MI is well known. As far back as 1994, it was demonstrated that thrombolysis within two hours halves the mortality rate for MI.[4] To demonstrate current thinking, Mr Ferguson quoted a Swedish cardiologist, Dr Leif Svensson:

> The infarcting heart can be likened to a burning building in our community: the longer the fire is left unchecked, the more damage it will do. At present the fire service arrive at the scene, load the burning building onto a truck and then take it back to the fire station to put the fire out. If we now have the opportunity to put the fire out at the scene of the catastrophe, we should certainly be doing this.

Thrombolytic agents that can be given as boluses, such as tenecteplase, are now available, making administration in the community more convenient. The Grampian region covers a large rural area with only one coronary care unit, in Aberdeen. General practitioners have been educated in cardiopulmonary resuscitation (CPR), defibrillation, electrocardiogram (ECG) interpretation and indications for thrombolysis. The GPs carry a 'cardiac pack', containing an ECG machine, IV access and a thrombolytic agent. For 'obvious' ECGs showing an MI, the GPs are happy to thrombolyse. If the GP is unsure about an ECG, the ambulance takes the patient to the nearest community hospital. Telemedicine facilities have been set up. Electrocardiograms can then be sent digitally to the Coronary Care Unit (CCU) in Aberdeen for a second opinion.

In Angus, the paramedics carry equipment that is capable of sending the ECG to the CCU in Dundee. If they arrive before the GP, they can then give thrombolysis on the advice of a cardiologist. This enables them to achieve first-contact-to-thrombolysis times of approximately 30 minutes.

Plans are underway in Grampian for troponin meters to be installed in community hospitals. This would enable low-risk patients with chest pain to be assessed in the community and avoid unnecessary emergency transfers to Aberdeen.

Rapid assessment and management of chest pain at the front door

Dr Alasdair Gray, Consultant in A&E Medicine, Royal Infirmary of Edinburgh

About 600,000 patients per annum attend A&E in the UK with chest pain: they represent 20–30% of the acute medical intake. Of these patients, 10% will have ST segment elevation, 25% will have an acute coronary syndrome, 20% will have an alternative diagnosis and 45% will have undifferentiated pain.[5]

History has a role in differentiating these patients. Radiation of pain to the jaw, shoulders and arms increases the likelihood of the pain being due to an MI. Reproduction of pain on exertion is a further useful discriminator. Unfortunately, 10% of patients with an MI describe the pain as burning, 10% as stabbing, and in 8% of patients it can be reproduced by palpation.[6]

Electrocardiograms are obviously helpful when they are abnormal, but a normal ECG is unhelpful in excluding an MI. Additional right and posterior leads[7] and serial ECGs[8] can increase the number of patients eligible for thrombolysis but are unhelpful in excluding an MI.

Various blood markers are widely used for the diagnosis of MI: troponin (T and I), creatinine kinase (total, mass, activity, subforms), myoglobin and combinations. None of these have a good negative predictive value for MI if taken within six hours of onset of chest pain,[9] see Figure 1.

Computer decision aids, combining the above information, have been shown to be effective in both diagnosing cardiac pain[10] and safely discharging patients.[11] The acute cardiac ischaemic time-insensitive predictive instrument has been shown to increase the number of patients safely discharged by 10–15%.[11]

Studies looking at patients discharged from A&E with chest pain show between 2% and 8% of acute myocardial damage has been missed. These patients then have a mortality rate that is twice as high as if they had been admitted.

Chest pain assessment units are common in the US. Low-risk patients with chest pain are observed for six to 12 hours with serial ECGs and blood markers. If negative, the patient then undergoes a provocation test such as an exercise tolerance test prior to discharge. Surveys of these centres suggest they are safe and lead to a reduced stay in hospital with a reduction in costs.[12] Six-hour rule-out protocols with ECGs and CK-MB mass[13] or CK-MB mass, troponin T, serial ECGs followed by an exercise tolerance test[14] have all been shown to be safe in the UK.

Currently, 10% of UK A&E departments have a low-risk pathway and 30% are planning one.[15] They are potentially safe, cost-effective and bed-saving.

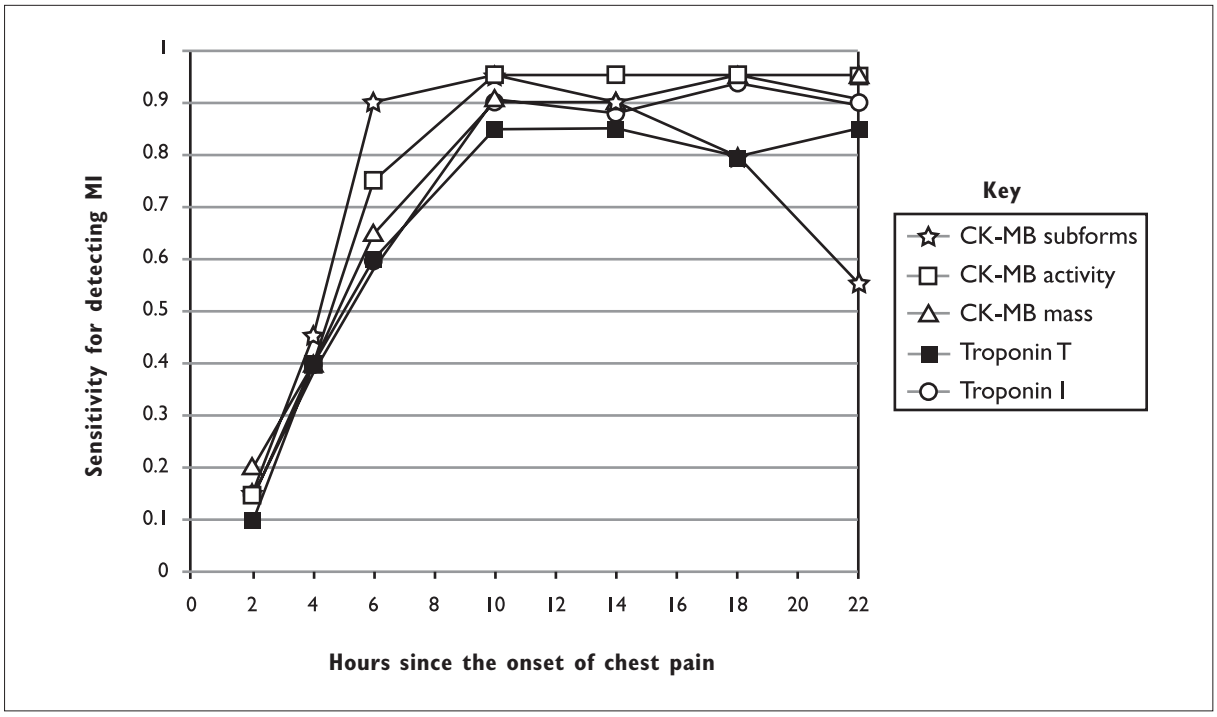

FIGURE 1
Commonly used blood markers for the diagnosis of MI.

Managing the high-risk patient

Professor Jennifer Adgey, Consultant Cardiologist, Royal Victoria Hospital, Belfast

An 'acute coronary syndrome' (ACS) includes unstable angina and non-Q-wave MI. Patients presenting with ACS are at high risk of MI or death. Patients who present with ST segment depression have a similar mortality rate at one month as those with ST segment elevation, and an increased mortality at three months.[16]

In unstable angina, a coronary artery plaque ruptures, resulting in platelet thrombosis and subsequent downstream emboli with minimal myocardial damage. The initial ECG is normal in 43% of patients, with T-wave inversion in 26% and ST segment depression in 20%.[17] Troponin is raised in one-third and indicates a high risk of MI or death in the following month.[18] Antman *et al.*[19] proposed seven risk score predictor variables: age ≥65 years; ≥3 coronary risk factors; prior coronary stenosis ≥50%; ST changes on initial ECG; ≥2 angina events in preceding 24 hours; aspirin taking in preceding seven days; and increased serum cardiac markers. More than three of these indicates a high risk.

High-risk patients need inhibition of arterial thrombogenesis. Three pathways of platelet activation can be inhibited: aspirin inhibits thromboxane, clopidogrel inhibits adenosine diphosphate and heparin inhibits thrombin. In addition, platelet aggregation can be directly inhibited by small molecule GP IIb/IIIa inhibitors. Low molecular weight heparin is superior to unfractionated heparin in preventing death, MI and urgent revascularisation. Clopidogrel has been shown to have an additive effect.[20] GP IIb/IIIa inhibitors have also been shown to be beneficial,[21] particularly in those patients who are likely to go on to angioplasty.

The FRISC II[22] study has shown that early invasive intervention in patients with ACS produces a 10·5% reduction in death or MI at 12 months. This effect was greatest for those patients with increased troponin levels. The TACTICS trial[23] compared early invasive intervention against intervention only in those patients with further pain or a positive exercise tolerance test. It showed a reduction in MI, death or re-hospitalisation from 24·2% to 14·3%.

Patients with ACS benefit from aspirin, nitrates, β-blockers, low molecular weight heparin, statins and clopidogrel. They require a stress test prior to, or soon after, discharge. High-risk patients, i.e. those with increased troponin, gain additional benefit from small molecule GP IIb/IIIa inhibitors and early angiography. See Figure 2.

After discharge: best community management

Dr Ewan Crawford, GP, Murrayfield Medical Centre, Edinburgh and Clinical Director, NW Edinburgh Health Care Co-operative

Cardiac rehabilitation programmes with education and psychological support lead to a 34% reduction in mortality, less depression and anxiety and fewer GP consultations. However, less than one-quarter of MI patients in Scotland receive formal rehabilitation. This can be subdivided into four phases.

Phase 1 is in-patient, phases 2 and 3 are immediate and

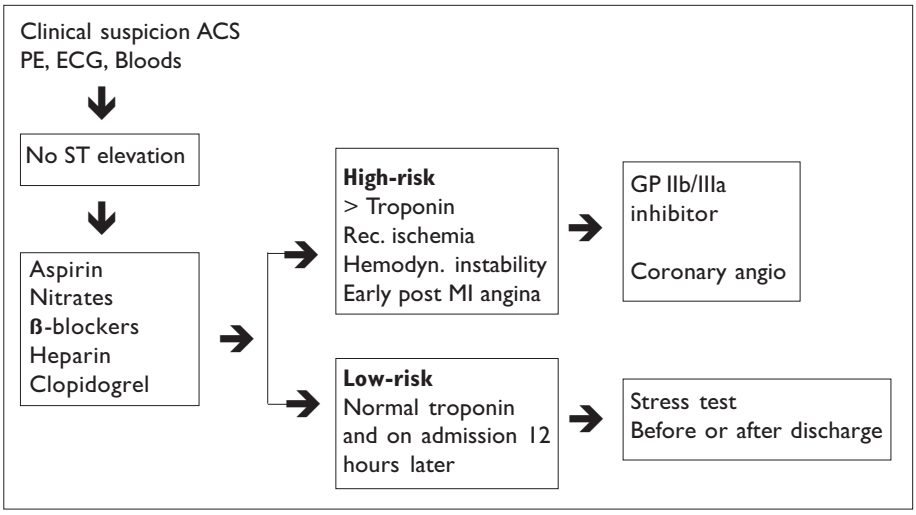

FIGURE 2
ESC Guidelines for UA/NSTEMI
(Source: *European Heart Journal* September 2000).

intermediate post-discharge and phase 4 is maintenance. Phases 1 and 2 involve explanation, reassurance and plan for the future. Phase 3 involves modifying risk factors (smoking, cholesterol, BP and weight), screening for diabetes and increasing exercise. Phase 4 involves maintaining behavioural changes and compliance with medications.

The Heart Manual is a Scottish Executive-backed programme. It is a six-week programme, involves a manual and tapes and is nurse-led. It addresses the issues in phases 2 and 3: exercise; smoking; anxiety/depression; relaxation; medication; and misconceptions. It tries to be individual to the patient's needs, encouraging patients to start from small beginnings and work up.

SESSION 3

MEDICAL EMERGENCIES UPDATE

Chaired by Dr Michael Jones, Consultant Physician, Ninewells Hospital and Medical School, Dundee

First fit: admit or not admit?

Dr Richard Davenport, Consultant Neurologist, Western General Hospital and Royal Infirmary of Edinburgh

Patients presenting with their first fit or convulsion require an accurate and rapid diagnosis, prognosis, investigations, treatment, advice and reassurance.

Differential diagnoses of patients presenting with altered states of consciousness include syncope, metabolic/endocrine conditions, toxic states (e.g. alcohol) and psychogenic problems. Epilepsy accounts for 30–40% of these patients presenting to A&E. A full history from the patient and witnesses is essential. This consists of circumstances, prodrome, event and recovery. Examination is usually unhelpful.

Dr Davenport shared some of his diagnostic tips:

- Generalised seizures always involve loss of consciousness.
- Syncope can cause myoclonic/tonic/clonic movements and occasionally reflex anoxic seizures.
- Transient ischaemic attacks never cause loss of consciousness.
- Urinary incontinence merely reflects whether the bladder was full when the patient lost consciousness.
- The scapula can be fractured during the tonic phase of seizure and if severe burns have occurred these indicate a deep level of unconsciousness.
- Biting of the side of the tongue or the inside of the mouth is also specific for seizures. Biting of the tip of the tongue merely indicates the tongue was protruding when the patient lost consciousness.
- After a generalised seizure patients will be drowsy, confused or aggressive for five to 30 minutes.
- Repeated episodes of unconsciousness lasting more than ten minutes are suggestive of psychogenic aetiology.

Having taken the history you may therefore be:

1. confident the patient has had an epileptic seizure;
2. confident of an alternative diagnosis; or (frequently when the event is unwitnessed)
3. uncertain.

If one is uncertain, a 'wait and see' approach is less harmful than an unwarranted diagnosis.

Investigations include a full blood count (FBC) immediately after the event, looking for neutrophilia; a white cell count exceeding 15 is suggestive of a seizure. Biochemistry and ECG are warranted if looking for alternative diagnoses. Specialist tests include electro-encephalogram (EEG) and brain imaging (CT (computerised tomography)/MRI). Electro-

encephalograms have a false negative rate of 30–50% and a false positive rate of 2%. Magnetic resonance imaging has a higher sensitivity than CT, with abnormalities picked up in 20–80% of patients. Most of these are minor atrophic abnormalities, with <10% having tumours. The SIGN guidelines[24] recommend EEGs for patients <25 years but no brain imaging if the diagnosis is idiopathic generalised epilepsy. For patients >25 years, brain imaging is recommended but EEGs only if the history is not clear.

Epilepsy cannot be diagnosed after a single seizure. For unprovoked first seizures, the two-year recurrence risk is 30–40%. Treatment is only indicated if there is an underlying structural lesion. However, all patients need advice and reassurance. Specialist nurses and support groups (Enlighten) can be helpful. All patients with an episode of loss of consciousness that is not vasovagal should be advised to inform the vehicle licensing agency. This is a legal duty and should be recorded in the notes. The impact of non-disclosure to the insurance company should be discussed with each patient.

If the patient has made a full recovery and there is nothing to suggest any underlying cause, they can be safely discharged from A&E. However, they should always be referred to see a specialist as an out-patient.

Safe management of diabetic emergencies

Dr Miles Fisher, Consultant Physician, Royal Infirmary, Glasgow

In this session, the management of hypoglycaemia, diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic state (HSS) were discussed. The Clinical Standards Board for Scotland[25] states that all patients who experience an acute diabetic emergency should be assessed rapidly and managed according to local protocols. The rate of diabetic emergencies should be monitored per patient. A specialist diabetes physician or nurse should review those admitted to hospital.

The American Diabetes Association recommendations[26] are based on two technical reviews.[27, 28] Diabetic ketoacidosis (defined as plasma glucose >14 mmol/L with H^+ >50, bicarbonate <15mmol/L and moderate ketonuria/ketonaemia) requires hospital admission. Hyperosmolar hyperglycaemic state (defined as impaired mental status, plasma osmolality >320 mmol/kg and glucose >33 mmol/L) also warrants hospital admission. It is recommended that patients with hypoglycaemia are admitted if:

1. blood glucose <2·8 mmol/L and treatment has not resulted in prompt recovery;
2. it has been complicated by coma, seizures or altered behaviour;
3. a responsible adult cannot be present for the ensuing 12 hours; or
4. if the hypoglycaemia is caused by a sulphonylurea.

Hypoglycaemia can be treated with oral ingestion of 20 g glucose, glucagon 1 mg s/c or i.m. or 25 g glucose i.v. Prior to discharge the cause should be identified and the risks of further hypoglycaemic episodes reduced by education, increased self-monitoring and increased professional support.

Management of DKA is based on the replacement of fluid, insulin and potassium. Inadequate potassium replacement is a preventable cause of death in these patients. Appropriate protocols should be available in A&E and acute wards, and adhered to. Prior to discharge patients require education, particularly in respect to increasing insulin when unwell. Improved communication with healthcare professionals can avoid admissions.

Managing variceal bleeding

Professor Roger Barton, Professor of Clinical Medicine, University of Newcastle

Professor Barton outlined the treatment of a patient with alcoholic liver disease presenting with haematemesis. If hypotensive, the patient requires resuscitation with fluids, and, as soon as possible, blood. Deficiencies in clotting factors should be corrected. The risk of re-bleeding is greatest in the first 48 hours and can be predicted by age, severity of shock (systolic BP <100 and PR >100) and co-morbidity (including the presence of liver failure).[29]

A central line should be inserted – if there is still significant haemodynamic disturbance after initial resuscitation – as should a urinary catheter. Drugs can be given to try to constrict the portal venous system. Terlipressin results in initial haemostasis in 70–90% of cases and reduces in-hospital mortality (10% vs 38%).[30] However, it is associated with cardiac complications and is often given with nitrates. Somatostatin[31] and octreotide[32] are also effective at inducing initial haemostasis and have fewer side-effects, but have never been shown to have any effect on survival.

Balloon tamponade is effective at temporarily controlling bleeding. However, intubation should be considered to reduce the risk of aspiration.

Immediate endoscopy can be attempted. Active bleeding can make it technically difficult. Sclerotherapy controls bleeding in >80% of cases, but severe complications are common (15%). Ligation may have a better prognosis[33] and has fewer complications, but is technically more demanding.

Transjugular intrahepatic portosystemic stent shunt (TIPSS) stops bleeding in 90% of cases. The major complication is hepatic encephalopathy. However, it does not influence survival in patients who have not responded to the above therapies. This is probably because these

patients have a very high mortality, perhaps as high as 100% at three months. Surgery such as transection, devascularisation or portal shunting has a mortality of 80% and should only be offered in very selected patients.

Infection is a common complication of bleeding – up to 66% – and may contribute to the poor prognosis. Prophylactic antibiotics, such as ciprofloxacin, increase survival by 9·1%.[34]

Professor Barton's recommendations are outlined in Figure 3.

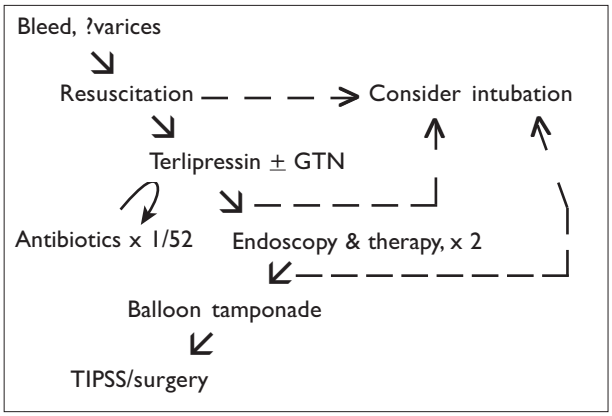

FIGURE 3
Recommendations for the emergency treatment of bleeding oesophageal varices.

Early management of acute renal failure

Dr Keith Simpson, Consultant Physician, Royal Infirmary, Glasgow

The incidence of acute renal failure (ARF) requiring renal replacement in Scotland is currently 215 per million per year: this is much higher than previously thought. About 52% of these patients are receiving their replacement therapy on ITU. On ITU, 76% of cases are due to acute tubular necrosis and 18% to pre-renal causes. On renal units the aetiology of the ARF is more varied.[35, 36]

Investigations of ARF are aimed at looking at the consequences (uraemia, potassium, sodium and water balance, anaemia) and the causes. History is the most helpful in identifying the cause, but in addition all patients need an ultrasound to exclude obstruction.

It has been standard practice to give frusemide and dopamine in acute oliguric renal failure. These drugs are not associated with any improvement in outcome[37, 38] and should only be used if diuretics or inotropes are indicated, and not as 'renal protection'.

Renal replacement therapy should be started when required, but there is no evidence to suggest the institution of early vs late dialysis influences the final outcome. The expensive biocompatible membranes do not improve the final outcome.[39] Continuous filtration is no better than intermittent dialysis.[40] However, if dialysis is used it needs to be daily;[41] if filtration is used it should be 35 ml/kg/hr or greater. Prognosis is poor in acute tubular necrosis (mortality 40–80%) because of co-morbidities.

CONCLUSION

The symposium was concluded with the Robert W. Philip Memorial Lecture. This was by Dr Mark Elliott and was entitled Managing Respiratory Failure in the Acute Setting. It will be highlighted in a forthcoming CD-ROM of this event due to be published in February 2003. The symposium was aimed at improving the care of patients with medical emergencies. It covered areas as diverse as new management techniques, improving waiting times and clinical guidelines for the management of variceal bleeding in order to do this.

REFERENCES

1 *Scottish Intercollegiate Working Party on Acute Medical Admissions and the Future of General Medicine.* Edinburgh: RCPE;1998. (www.rcpe.ac.uk/publications/articles/acute.html)
2 *Acute Medicine the Physicians Role. Proposals for the Future: A working party report of the Federation of Medical Royal Colleges.* London: RCPL; 2000.(www.rcplondon.ac.uk/pubs/wp_acute)
3 *Governance in Acute General Medicine*. London: Royal College of Physicians; 2002 (Nov).
4 Boersma E, Maas AC, Deckers JW *et al.* Early thrombolytic treatment in acute myocardial infarction: reappraisal of the golden hour. *Lancet* 1996; **348(9030):**771–5.
5 Lee TH, Goldman L. Evaluation of the patient with acute chest pain. *N Engl J Med* 2000; **342(16):**1187–95.
6 Lee TH, Cook EF, Weisberg M *et al.* Acute chest pain in the emergency room. Identification and examination of low-risk patients. *Arch Intern Med* 1985; **145(1):**65–9.
7 Sgarbossa EB, Birnbaum Y, Parrillo JE. Electrocardiographic diagnosis of acute myocardial infarction: Current concepts for the clinician. *Am Heart J* 2001; **141(4):**507–17.
8 Fesmire FM, Percy RF, Bardoner JB *et al.* Usefulness of automated serial 12-lead ECG monitoring during the initial emergency department evaluation of patients with chest pain. *Ann Emerg Med* 1998; **31(1):**3–11.
9 Hamm CW. Cardiac biomarkers for rapid evaluation of chest pain. *Circulation* 2001; **104(13):**1454–6.
10 Goldman L, Cook EF, Johnson PA *et al.* Prediction of the need for intensive care in patients who come to the emergency departments with acute chest pain. *N Engl J Med* 1996; **334(23):**1498–504.
11 Selker HP, Beshansky JR, Griffith JL *et al.* Use of the acute cardiac ischemia time-insensitive predictive instrument (ACI-TIPI) to assist with triage of patients with chest pain or other symptoms suggestive of acute cardiac ischemia. A multicenter, controlled clinical trial. *Ann Intern Med* 1998; **129(11):**845–55.
12 Zalenski RJ, Rydman RJ, Ting S *et al.* A national survey of emergency department chest pain centers in the United States. *Am J Cardiol* 1998; **81(11):**1305–09.
13 Herren KR, Mackway-Jones K, Richards CR *et al.* Is it possible to exclude a diagnosis of myocardial damage

within six hours of admission to an emergency department? Diagnostic cohort study. *BMJ* 2001; **323(7309):**372.
14 Goodacre SW, Morris FM, Campbell S *et al.* A prospective, observational study of a chest pain observation unit in a British hospital. *Emerg Med J* 2002; **19(2):**117–21.
15 Goodacre SW *et al.* National survey of emergency department management of patients with acute undifferentiated chest pain. Unpublished.
16 Savonitto S, Ardissino D, Granger CB *et al.* Prognostic value of the admission electrocardiogram in acute coronary syndromes. *JAMA* 1999; **281(8):**707–13.
17 Hamm CW, Goldmann BU, Heeschen C *et al.* Emergency room triage of patients with acute chest pain by means of rapid testing for cardiac troponin T or troponin I. *N Engl J Med* 1997; **337(23):**1648–53.
18 Ottani F, Galvani M, Ferrini D *et al.* Direct comparison of early elevations of cardiac troponin T and I in patients with clinical unstable angina. *Am Heart J* 1999; **137(2):**284–91.
19 Antman EM, Cohen M, Bernink PJ *et al.* The TIMI risk score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. *JAMA* 2000; **284(7):**835–42.
20 Yusuf S, Zhao F, Mehta SR *et al.* Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. *N Engl J Med* 2001; **345(7):**494–502.
21 Boersma E, Harrington RA, Moliterno DJ *et al.* Platelet glycoprotein IIb/IIIa inhibitors in acute coronary syndromes: a meta-analysis of all major randomised clinical trials. *Lancet* 2002; **359(9302):**189–98.
22 Wallentin L, Lagerqvist B, Husted S *et al.* Outcome at 1 year after an invasive compared with a non-invasive strategy in unstable coronary-artery disease: the FRISC II invasive randomised trial. FRISC II investigators. Fast revascularisation during instability in coronary artery disease. *Lancet* 2000; **356(9223):**9–16.
23 Cannon CP, Weintraub WS, Demopoulos LA *et al.* Comparison of early invasive and conservative strategies in patients with unstable coronary syndromes treated with the glycoprotein IIb/IIIa inhibitor tirofiban. *N Engl J Med* 2001; **344(25):**1879–87.
24 Scottish Intercollegiate Guidelines Network. *SIGN: 21. Diagnosis and Management of Epilepsy in Adults*. Edinburgh: SIGN; 1997.
25 *Clinical Standards: Diabetes*. 2nd edition. Edinburgh: Clinical Standards Board for Scotland; 2002.
26 American Diabetes Association: clinical practice recommendations 2002. *Diabetes Care* 2002; **25(Suppl 1):**S1–147.
27 Cryer PE, Fisher JN, Shamoon H. Hypoglycemia. *Diabetes Care* 1994; **17(7):**734–55.
28 Kitabchi AE, Umpierrez GE, Murphy MB *et al.* Management of hyperglycemic crises in patients with diabetes. *Diabetes Care* 2001; **24(1):**131–53.
29 Rockall TA, Logan RF, Devlin HB *et al.* Risk assessment after acute upper gastrointestinal haemorrhage. *Gut* 1996; **38(3):**316–21.
30 Levacher S, Letoumelin P, Pateron D *et al*. Early administration of terlipressin plus glyceryl trinitrate to control active upper gastrointestinal bleeding in cirrhotic patients. *Lancet* 1995; **346(8979):**865–8.
31 Feu F, Ruiz del Arbol L, Banares R *et al.* Double-blind randomized controlled trial comparing terlipressin and somatostatin for acute variceal hemorrhage. Variceal Bleeding Study Group. *Gastroenterology* 1996; **111(5):**1291–9.
32 Gotzsche PC. Somatostatin or octreotide for acute bleeding oesophageal varices. *Cochrane Database Syst Rev* 2000; **(2):**CD000193.
33 Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. *Ann Intern Med* 1995; **123(4):**280–7.
34 Bernard B, Grange JD, Khac EN *et al.* Antibiotic prophylaxis for the prevention of bacterial infections in cirrhotic patients with gastrointestinal bleeding: a meta-analysis. *Hepatology* 1999; **29(6):**1655–61.
35 Feest TG, Round A, Hamad S. Incidence of severe acute renal failure in adults: results of a community-based study. *BMJ* 1993; **306(6876):**481–3.
36 Liano F, Pascual J. Epidemiology of acute renal failure: a prospective, multicenter, community-based study. Madrid Acute Renal Failure Study Group. *Kidney Int* 1996; **50(3):**811–18.
37 Shilliday IR, Quinn KJ, Allison ME. Loop diuretics in the management of acute renal failure: a prospective, double-blind, placebo-controlled, randomized study. *Nephrol Dial Transplant* 1997; **12(12):**2592–6.
38 Bellomo R, Chapman M, Finfer S *et al.* Low-dose dopamine in patients with early renal dysfunction: a placebo-controlled randomised trial. Australian and New Zealand Intensive Care Society (ANZICS) Clinical Trials Group. *Lancet* 2000; **356(9248):**2139–43.
39 Jorres A, Gahl GM, Dobis C *et al.* Haemodialysis-membrane biocompatibility and mortality of patients with dialysis-dependent acute renal failure: a prospective randomised multicentre trial. International Multicentre Study Group. *Lancet* 1999; **354(9187):**1337–41.
40 Mehta RL, McDonald B, Gabbai FB *et al.* A randomized clinical trial of continuous versus intermittent dialysis for acute renal failure. *Kidney Int* 2001; **60(3):**1154–63.
41 Schiffl H, Lang SM, Fischer R. Daily hemodialysis and the outcome of acute renal failure. *N Engl J Med* 2002; **346(5):**305–10.